Fat Embolism Syndrome

A case presentation

Thanh Nga Tran, Harvard Medical School, Year IV
Gillian Lieberman, MD
Our Patient

- H.K is a 58 year-old woman with a history of afib on amiodarone, HTN, CAD, DM, high chol, s/p gastric bypass presented 2/3/2005 after a mechanical fall.
- Pt slipped on hardwood floor. Had right distal femur comminuted fracture.
- She was admitted to the orthopedics service, → OR on 2/4/2005 for ORIF of her right femur.
- Pre-operatively, pt had intact mental status and moved all extremities well.
Comminuted fracture of the distal right femur with approximately 5 mm of foreshortening, and anterior distraction.

Calcifications in distal femur & proximal tibia: Evidence of old endochondromas
Enchondroma: Benign, no periosteal rxn, central, characteristic ring and arc calcification

Ddx in long bone: bone infarction

In association with pathologic fx → likely enchondroma
Intra-operative Course

Partway through, our patient acutely decompensated with SBP 60s-70s, hypoxia to 80s, end tidal CO$_2$ 37.

She was started on pressors, first dopamine, then neosynephrine, then epinephrine.
Ddx of Acute Hypoxia

- Pulmonary embolism from:
  - Clot
  - Fat
  - Air

- Pneumothorax
- Acute MI
- Mechanical: ET tube, machine malfunction
Menu of Tests Available for Work-up

Concern for massive PE usual work-up may include:

- CXR
- ABGs
- Hemodynamic monitoring
- Echocardiogram
- V/Q scan
- Pulmonary angiogram – gold standard
- Helical CT – PE protocol
- MRA

But patient was in the O.R.
Intra-Operative TEE

- Emergent intra-op Transesophageal Echocardiogram (TEE) revealed dilated RV cavity size with RV free wall hypokinesis, mild pulmonary hypertension, new PFO concerning for massive PE.
- Pt was given herparin initially.

Emergent intra-op pulmonary arteriogram was performed.
Intra-Operative Pulmonary Arteriogram

- No firm centrally-occlusive clot.
- Markedly decreased flow in both pulmonary arteries & no central filling defects, compatible with markedly ↑↑ micro-circulatory vascular resistance.
Ddx includes fat embolism, air embolism, possibly extremely soft clot.

Pt received Nitric Oxide (NO) for ?vasospasm with some improvement to flow

- Two pts w massive PE: condition only improved after administration of NO

Pt received intra-arterial tPA to both arteries for clot prevention

IVC filter placed
Post-Operative Course

- Pt noted to have flaccid paresis post OR and next day → onset of L hemiparesis
- Stat head CT to r/o intracranial hemorrhage, brain infarction
Anatomic Review of Arterial Supply to Brain

Blood supply to the brain-color coded

- Anterior cerebral (orange)
- Middle cerebral (pink)
- Posterior cerebral (blue)

www.stroke-information.net/allcerebsupply.htm
Our Patient Head CT

- B/l cerebellar infarcts
- B/l occipital (PCA territory) infarcts
- Temporal infarct
- Diffuse hypodensity in white matter
Our Patient Head CT

Brainstem infarction

Punctated hemorrhages

PACS, BIDMC
Results of Head CT

- Bilateral occipital and cerebellar infarcts, & diffuse hypodensity within white mater & brainstem, likely from fat emboli.
- Punctate hypodensities w/in the R temporal lobe → ?focal areas of petechial hemorrhage?
- Recommend MRI
Our Patient MRI

- **T1** – Image anatomy – fluid dark
- **T2** – Image pathology – fluid bright
- **FLAIR** (Fluid-Attenuated Inversion Recovery) – Image brain pathology
  - Edema, inflammation
  - T2 w fluid suppressed
- **Diffusion** – sensitive for acute stroke (~ <2hrs)
Our Patient Flair/Diffusion
Our Patient Flair/Diffusion
Our Patient Flair/Diffusion
Results of MRI

- There are multiple areas of increased T2/FLAIR/DWI signal in L occipital, temporal, frontal, deep thalamic, and R temporal, occipital lobes, & R cerebellar hemisphere c/w recent infarction.

- Increased T2/FLAIR signal is also visualized in the pons and L midbrain, which does not demonstrate increased signal on the DWI imaging sequence.

- Brain edema is visualized in these areas of infarction.
Review of Vascular Anatomy

CIRCLE OF WILLIS

- Internal Carotid Artery
- Anterior Cerebral Artery
- Circle of Willis
- Anterior Communicating Artery
- Middle Cerebral Artery
- Posterior Communicating Artery
- Basilar Artery
- Posterior Cerebral Artery
- Vertebral Artery
- Posterior Inferior Cerebellar Artery

www.csuchico.edu/.../CMSD%20320/362unit11.html

www.southeastiowaopenmri.com/Clinical_Photos.htm

www.gehealthcare.com/.../os_images.html
Signal loss in the distal portion of the left PCA, consistent with the territorial infarction seen on the brain MR. The rest of the major tributaries of the circle of Willis are patent.
Our Patient Course

- EEG on 2/20/05 2/2 to R-sided weakness and inability to tolerate head CT: non-convulsive status epilepticus
- Repeat head CT
New L hemorrhagic conversion CVA w/in L occipital, temporal and parietal region
Several regions of stable, evolving infarction and hemorrhagic conversion: L occipital, R right parietal, R temporal lobe; and b/l frontal lobe infarctions.
Our Patient Course

- Pt is s/p ORIF w fat emboli to lungs, brain through PFO, hemorrhagic conversion of CVA and subsequent non-convulsive status epilepticus (treated).
- Pt also had NSTEMI, line sepsis, respiratory failure and hypotension (could be 2/2 to aspiration pneumonitis or PNA), and acute renal failure.
- Pt is non-responsive, occasional grimace.
- Pt currently still in MICU. Supportive care given to family for the tragic complication.
Fat Embolism Syndrome (FES)

- First described by Zenker and later Von Bergmann in mid 1800s
- Mortality 5-15%
- Frequently assoc with long bone/pelvic fractures, more frequent w/ closed fx; orthopedics surgeries, liposuction, burns, soft tissue injuries
- Non-trauma: pancreatitis, DM, osteomyelitis, bone tumor lysis, steroids, fatty liver, cyclosporin, lipid infusion
- FES typically manifests 12 to 72 hours after the initial insult or as late as two weeks
FES

- **Classic triad**: hypoxemia; neurologic abnormalities; and a petechial rash (pathognomonic)

- **Early findings**: dyspnea, tachypnea, and hypoxemia

- **Neurological abnl often followed**: confusional state, delta MS, occasional seizures and focal deficits

- **Late**: possible petechial rash of head, neck, anterior thorax, subconjunctiva, and axillae – resolved in 5-7 days

- **Minor**: lipiduria, scotomata (Purtscher's retinopathy), fever, coag abnl, myocardial depression: 2/2 to toxic mediators or dysfunctional lipid metabolism
### Table 1 Signs and Symptoms of the Fat Embolism Syndrome

<table>
<thead>
<tr>
<th>Major criteria</th>
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<tr>
<td>Petechial rash</td>
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<td>Respiratory symptoms with diffuse, bilateral, patchy alveolar or interstitial infiltrates on chest radiography</td>
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<td>Neurologic changes not explained by trauma or other medical condition</td>
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<tr>
<td>Hypoxemia</td>
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<td>Minor criteria</td>
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<tr>
<td>Tachycardia</td>
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<td>Fever</td>
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<td>Funduscopic examination findings (cotton-wool spots or fat droplets in arterioles)</td>
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<td>Diminished urine output or fat globules</td>
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<tr>
<td>Decreasing hematocrit</td>
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<td>Thrombocytopenia</td>
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<td>Elevated erythrocyte sedimentation rate</td>
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<td>Fat globules in sputum</td>
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Gurd credited with scoring system

FES Etiology

**Mechanical:** Occlusion of blood vessels by fat globules from trauma (marrow, adipose)- With continued embolization, pulmonary artery and right heart pressures rise → PFO → systemic circulation → paradoxical embolism.

**Biochemical:** Via toxic intermediates of plasma-derived fat such as chylomicrons or infused lipids: embolized fat → free fatty acids (FFA) → directly toxic to pneumocytes and capillary endothelium in the lung, causing interstitial hemorrhage, edema and chemical pneumonitis; also cardiac contractile dysfx.
Diagnosis

- Clinical presentation

- Radiologic:
  - **CXR**: nl in most, minority w evenly distributed, fleck-like pulmonary shadows (*Snow Storm* appearance), increased pulm markings and R heart dilatation
  - **V/Q**: mottled pattern of subsegmental perfusion defects with a normal ventilatory pattern
  - **Chest CT**: Focal areas of ground glass opacification with interloblar septal thickening
  - **Brain MRI**: may reveal high intensity T2 signal, mult scattered lesions in white mater & ischemia on DWI & FLAIR
  - **PET scanning**: cerebral blood flow alterations & correction

- No reliable lab tests
Starfield pattern of scattered white spots pathognomonic for acute microinfarcts. High intensity = edema

Ddx: diffuse axonal injury, edema w microinfarcts, gliosis, demyelinating disease

Parizel et al Stroke. 2001, p 2042
Multiple focal lesions in periventricular, deep, subcortical white matter – characteristic of cerebral FE

Demetriads et al Arch Surg 2004; 139, 1257

Simon et al AJNR 2003; 24:97-101
Radiology FES - MRI

Patient 5: Post-traumatic paradoxical fat embolism to brain

Patient 6 - 24 yo M w comminuted femur fx: Geographic appearance of diffuse ground-glass opacities

Patient 7 - 19 yo M 2 days after femur fx: ground glass opacities w smooth & nodular septal thickening

Malagari et al Chest 2003, 1196
Treatment

- Early immobilization of fractures
- Operative correction
- Supportive care
  - Maintain of intravascular volume as shock can exacerbate the lung injury. Recs albumin binds fatty acids, and may decrease the extent of lung injury
  - Mechanical ventilation and PEEP may be required to maintain PaO2.
  - High dose corticosteroids have been effective in preventing development of FES in several trials, but controversy still persists
Other Unusual Embolisms
Amniotic Fluid

- **Patient 8**: 29 yo G1P1 – still birth 3mo prior, p/w R thorax dullness. CXR \(\rightarrow\) c/w cystic lung mass

- CT – homogeneous, w air, hyperdense border, density similar to pleural effusions

Kaptanoguu *et al* Scand Cardiovasc 1999, 117
Atrial Myxoma

- **Patient 9**: 58 yo F p/w syncope

- **2-D TTE**: Giant myxoma in the left atrium (A). The left atrial myxoma going through the mitral valve into the left ventricle during diastole (B).

Acikel *et al* Int J Card 2004, 325
Myxoma Emboli

Patient 10: 50 yo F w transient L arm weakness and L atrial mass c/w myxoma & meningeal metastasis

Resection: low grade myxosarcoma

Hirudayaraj et al Int J Card 2004, 471
**Air Emboli**

- **Patient 11:** 60 yo M w cough, wt loss, DOE s/p Transbronchioscopy Lung biopsy
- **Fig. 1:** Well-defined, rounded foci of hypodensity in the left high frontoparietal regions suggestive of air embolism.
- **Fig. 2:** Three weeks later – b/l watershed ANCA / MCA and R MCA/PCA territory infarcts (arrows).

Shetty, P et al. Australasian radiology, 2001; 215
**Air Emboli**

**Patient 12:** 35 yo M s/p lung resection for aspergilloma, tension pneumothorax s/p chest tube → sudden delta MS, L face, arm, leg weakness

- CT: hypointensity in R MCA c/w air
- MRI – DWI – increased signal intensity in R MCA territory

**Patient 13:** a more dramatic case of air embolism

_Hodics et al Neurology 2003; 60-112_
Methacrylate Emboli

- Patient 14: 54 yo M s/p vertebroplasty for mets esophageal CA with abnl CXR
- CT: Mid to distal pulmonary vessels → dense material c/w metacrylate emboli.

Distal to the metacrylate: the pulmonary vessels are low density c/w lack of perfusion.
References

- Up-to-date: [http://www.uptodate.org](http://www.uptodate.org)
- Odegard, K. Fat Embolism: Diagnosis and Treatment. [http://www.orthoteers.co.uk/Nrujp-ij33lm/Orthfatembolism.htm](http://www.orthoteers.co.uk/Nrujp-ij33lm/Orthfatembolism.htm)
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References

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